UNITED STATES DISTRICT COURT
DISTRICT OF MINNESOTA

IN RE NATIONAL HOCKEY PLAYERS' CONCUSSION INJURY LITIGATION

Court File No. 14-md-2551 (SRN-JSM)

REBUTTAL DECLARATION OF THOMAS BLAINE HOSHIZAKI, PH.D

This Document Relates to:

ALL ACTIONS

1. My name is Thomas Blaine Hoshizaki. I am submitting this Rebuttal Declaration as a supplement and clarification to the Declaration of Thomas Blaine Hoshizaki, Ph.D that was filed in the above-referenced matter as ECF Doc. No. 645 ("Initial Declaration").

2. I understand that Defendant National Hockey League ("NHL") has hired a number of people who have criticized aspects of my Initial Declaration and the facts and opinions in it. This declaration is intended to respond to certain of those criticisms.

3. Many of the criticisms that have been levied concerning the Initial Declaration are descriptions of limitations in measuring head impact frequency of NHL players. There are limitations in the ability to make numerous measurements, including the frequency of head impacts in National Hockey League players. However, the mere presence of limitations does not render my methodology or conclusions incorrect, so long as the limitations are acknowledged and my conclusions can be verified by the conclusions

in other research. I have acknowledged limitations in my analysis and conclusions. In addition, my conclusions are verifiable as further set forth in this document. Furthermore, as set forth in more detail in the remainder of this Declaration, my methodology was conservative in many aspects.

Validity of Measurements

4. The validity and reliability of my Initial Declaration, including its ultimate conclusions, are confirmed through comparison to the methodologies and conclusions of others. Those comparisons, as further described in this Declaration, demonstrate that the data set that was described in my Initial Declaration concerning the frequency of hits, the magnitude of hits, and the resulting strain is correct; in fact, if anything it demonstrates that my figures are conservative.

5. As to my ultimate conclusions concerning head impacts to NHL players, they are verifiable by comparison to other data sets:

a. As to the number of head impacts (frequency), that conclusion is comparable to the NHL's own video study. The NHL's own video study measured a slightly lower frequency, but their assessment was proximately close to my own findings:

Season	**NHL Head Impacts Per Game**	**Hoshizaki Head Impacts Per Game**
1986-1987	N/A	22.7
1995-1996	N/A	28
2003-2004	N/A	47.7
2005-2006	32	N/A
2006-2007	36	N/A
2007-2008	38	N/A
2008-2009	42	N/A
2009-2010	44	N/A
2010-2011	45	N/A
2011-2012	45	N/A
2013-2014	N/A	55.7

The head impacts per game are close; the minor differences between the two counts are attributable to different games analyzed and methods of categorization. The head impact frequencies are also consistent with the impacts reported in the NHL report, even though the games reviewed in that study were not the same games reviewed in my analysis. Whether one or two impacts were included or excluded does not materially affect the opinion in the Initial Declaration or this Declaration. This in and of itself provides an unbiased and objective confirmation that the methods used and frequency values are accurate. Not only is this the NHL's own conclusion, but the NHL's analysis was based on 1,800 games, which is a significantly higher number of games than that which was analyzed to produce the facts and conclusions in the Initial Declaration.

b. Moreover, the other manner in which the number of head impacts (frequency) can be measured, other than by video analysis, is by using accelerometers.[1] Using video to measure head impact frequency requires cameras to capture the event, and therefore fails to account for those hits that occur off-camera (another reason why the frequency measured is conservative). A second measure for documenting head frequency is using accelerometers in hockey helmets, which measure the frequency and magnitude of athletes, but therefore capture a wider range of impacts than we measured (*e.g.* contact with the goal, contact with the stick, indirect contact, and contact from celebrating). Fifteen to twenty percent of impacts recorded when using accelerometers are not captured on video.[2] Use of accelerometers provides a frequency rate that is much *higher* than that

[1] Jadischke, *et al.*, *On the accuracy of the Head Impact Telemetry (HIT) System used in football helmets*, 46 JOURNAL OF BIOMECHANICS, 2310-15 (2013).

[2] Wilcox BJ, *et al.*, *Head-Impact Mechanisms in Men's and Women's Collegiate Ice Hockey*, 49(4) JOURNAL OF ATHLETIC TRAINING 514-520 (2014).

observed through my own video analysis demonstrating that if anything, my conclusions are conservative.

c. The frequency of head impacts in an NHL ice hockey game as set forth in my Initial Declaration were not exaggerated or inaccurate and are supported by both published helmet accelerometer data and the NHL's own head impact data from video analysis.

d. As to the magnitude of the impacts (velocity), those results were compared to data set forth in scientific literature from others,[3] which data demonstrated that if anything, the results of my research were conservative as to the magnitude. While there are limitations as to methods of measuring impact velocities, the impact values that I derived through my analysis are comparatively low considering what we know about the velocity at which people can skate, punch, or fall to the ground.

e. As to the MPS values, the results of my research were comparable to the values reached in literature from others, if not lower.[4] This comparison also demonstrates that the results of my research were conservative, if anything.

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[3] Roy B. and Doré R., *Kinematics of the Slap Shot in Ice Hockey as Executed by Players of Different Age Classifications*, 5TH INTERNATIONAL CONGRESS OF BIOMECHANICS, International Congress of Biomechanics, Jyväsklä, Finland, June 29 – July 7, 1975; Pearsall, D. J., Montgomery, N., and Turcotte, R. A., *The Influence of Stick Stiffness on the Performance of Ice Hockey Slap Shots*, 2 SPORTS ENG., 3-11 (1999); Rousseau, P., Hoshizaki, T. B., and Gilchrist, M. D., *For ASTM F-08: Protective Capacity of Ice Hockey Player Helmets against Puck Impacts*, MECHANISM OF CONCUSSION IN SPORTS, STP 1552, Alan Ashare and Mariusz Ziejewski, Eds., pp. 196-207, doi:10.1520/STP155220120159, ASTM International, West Conshochocken, PA 2014; Rousseau, P., Post, A., and Hoshizaki, T. B., *A Comparison of Peak Linear and Angular Headform Accelerations Using Ice Hockey Helmets*, J. ASTM INT., Vol. 6 (2006), Paper ID JA101877.

[4] Kleiven S., *Predictors for traumatic brain injuries evaluated through accident reconstruction*, 51 STAPP CAR CRASH J, 1-35 (2007); Patton D., McIntosh A., Kleiven S., *The Biomechanical Determinants of Concussion: Finite Element Simulations to Investigate Brain Tissue Deformations During Sporting Impacts to the Unprotected Head*, 29 JOURNAL OF APPLIED BIOMECHANICS, 721-730 (2013); Zhang L, Yang KH, King AI, *A proposed injury threshold for mild traumatic brain injury*, 12 J BIOMECH ENGINEER, 226-

f. In fact, if my laboratory hadn't conducted any video analysis or issued any report of facts and conclusions, but instead used the facts and figures set forth in comparative literature and studies, the numbers for frequency, velocity, and strain from head impacts would have been higher.

6. Individuals hired by the NHL criticized my findings and conclusions claiming that post-impact motion was not taken into account and therefore the velocity could not be accurately measured. However, my reconstruction of the impacts observed on the video did account for post-impact velocity. Specifically:

a. The model used in the reconstructions used a certified ice hockey helmet on an instrumented Hybrid III head form attached to a compliant unbiased neck form that is in turn attached to a 40 kg sliding table. When the helmeted head form is impacted, the compliant neck and sliding table allow the head to move out of the way of the impacting object in this way replicating the change in velocity of the impactor during the actual impact. As a result, the impacting object is able to maintain post-impact velocity. The head of the model is impacted using the location and direction of impact, therefore accounting for direct and glancing blows. When a glancing blow occurs in real life, the decrease in velocity pre and post is not as great as a direct blow; the glancing blow is duplicated in the recreation of the event in the lab allowing the impacting object to maintain post-impact velocity as would happen in real life. In conditions where the helmeted head impacts the glass, boards, or wall, those elements move very little or not at all and on glancing impacts, the helmeted head continues to move along the glass, boards and ice. These events were reconstructed to replicate the live events documented in the NHL game videos.

236 (2004); Deck C, Willinger R, *Improved head injury criteria based on head FE model*, 13(8) INTERNATIONAL JOURNAL OF CRASHWORTHINESS, 667-78 (2008).

b. I acknowledge that when the head is impacted, the impacting body will continue to have energy. That is why the reconstruction allows the impactor to continue after its contact with the head.

c. The compliance of the helmeted head in the reconstruction is accounted for by the compliance of: the Hybrid III head form, certified hockey helmet(s), ice, glass, boards, elbows, shoulders, and puck. For the ice, an actual ice surface was used to reconstruct the ice impact, as was a poly carbonate sheet for the glass and wood to represent the boards. For the puck impact, an ice hockey puck was used as the impacting object. For the shoulder impacts, we used a compliant impactor that matched the compliance or elasticity of a padded ice hockey player's shoulder; the compliance was determined by having ice hockey players impact the helmeted Hybrid III head with their shoulder and ice hockey shoulder pad, creating an acceleration curve that was then duplicated using vinyl nitrile foam to ensure the shoulder impactor matched the compliance of a player's padded shoulder. For the elbow, hockey players impacted the helmeted Hybrid III head with their elbow and ice hockey elbow pad creating an acceleration curve that was then duplicated using vinyl nitrile foam to ensure the elbow impactor matched the compliance of a player's padded elbow. In this way, the head impact reconstructions included the appropriate elastic characteristics for each head impact reconstructed in this report.

d. The manner in which the reconstruction is created accounts for the elements that matter—speed, mass, direction and location.

e. To the extent that the NHL's critique concerning post-impact velocity is the claim that my analysis should have measured post-impact velocity of the struck player in the video and ensured that the head form matched it, that is a manner of confirming the reconstruction, but the head form used is already validated for appropriate biodynamic response of the head making such a check unnecessary.

7. A further criticism from those individuals retained by the NHL was a claim that the reconstructions in my laboratory failed to account for elasticity, and instead only demonstrated an impact on the impacted player, as opposed to both collision partners. As set forth in the preceding paragraph, I did account for the collision impact to both partners. I used an "elastic impactor" to account for the elasticity of the impacting element (*e.g.* the shoulder). The elastic impactor mimics the elasticity of the athlete's shoulder and shoulder pad.

8. One of the criticisms from those retained by the NHL is that the strain that was used to determine the thresholds for arriving at the logical conclusions of the risk of concussion, was maximum principal strain ("MPS"), as opposed to axonal strain. Those individuals go on to suggest that a measurement of "axonal strain" is necessary to accurately diagnose the risk of concussion and suggest that the same percentage threshold for risk of concussion should be used but with axonal strain percentages applied to that threshold (this is called an anisotropic model). There are numerous problems with the approach advocated by the individuals hired by the NHL concerning their criticisms related to axonal strain measurement:

a. I used MPS as the measurement of the risk of a concussion from impact to the head because MPS has a known and demonstrated ability to measure trauma.
b. I acknowledge that MPS is the strain on an element and not the strain along an axon. The MPS models that I have presented in my analysis, including in my Initial Declaration, are not intended to represent strain thresholds for axons; rather, they are intended to measure brain tissue strain.
c. The literature shows that MPS strain can be the same as axonal strain; it simply depends on if the two strains are going in the same direction. We know, even according to charts in the reports of the individuals retained by

the NHL, that they acknowledge that MPS values are associated with the concept of axonal strain.[5]

d. Using an anisotropic model, as suggested by certain of the individuals retained by the NHL, has at least two problems:

i. There is no data set for the anisotropic model to use for comparison to determine if the results of an anisotropic model are valid. As such, an anisotropic model is theoretical and not validated. The best that a finite element anisotropic model would be able to do is to model the effect of axonal tracks, which might result in lower values, but it's unknown whether those lower values represent actual axial injury because there is little reference data, partial or otherwise. In contrast, my laboratory has more than 600 real data points—with known injury outcomes—against which to compare the results using MPS. Even if in theory, a more complex model could produce more accurate results, there is no way to interpret the results without reference data sets. Given the dearth of reference data to support the use of an anisotropic model, the use of MPS values rather than the measure of axonal strain provides in my opinion far more relevant results.

ii. The suggestion that the magnitude of the impacts is too high as a result of not using an anisotropic model is also without merit. The scientific literature provides data on the threshold levels of *MPS* that result in concussions, a data point that we don't have in the case of an anisotropic model.[6]

9. At least one of the individuals hired by the NHL to criticize my Initial Declaration and conclusions in this matter – Dr. Matthew Panzer – offered a sensationalized claim that the model I employed in arriving at my factual findings and

[5] Declaration of Matthew Panzer [ECF Doc. 732-10] at 54, Fig. 15.

[6] *See supra* n. 4.

conclusions would result in every person who has ever jump-roped having experienced a permanent brain injury. There are numerous issues with this claim.

a. First and foremost, I have never claimed that permanent "brain injury" occurs if there is 5-8% MPS. (*See infra* the "Scope of Conclusions" section of this declaration.)

b. Second, the numbers that were used to arrive at Dr. Panzer's assertions are inherently flawed, as evidenced by dynamic response values being incredibly low compared with the resulting claimed MPS values. For example, in the course of years of studying this subject I have never heard of, much less seen, an impact at 4 g and 200 rad/s^2 result in 45% MPS. In completing hundreds of impact reconstructions using the UCD model, I have never observed MPS values associated with the extremely low acceleration values that are assigned to these activities. To my knowledge, there is no published or unpublished data that is consistent with the MPS values that the NHL's hired consultant reports for the events of "head slap," "chair plop," "head shake," and "skipping rope." These accounts by Dr. Panzer are particularly problematic considering the reported kinematic variables that Dr. Panzer used to calculate MPS. If using the NHL's claimed MPS values comparatively, it's clear that, if anything, the numbers in my conclusion were low, considering:

    i. Dr. Panzer reports a "head shake" creates a 4g peak linear acceleration and 200 rad/s^2 peak rotational acceleration, all of which he estimates to result in 45% MPS when using my method of analysis. In my report, there were three examples of 41%-43% MPS:

        1. a high velocity punch to the head (2013-14 season), but to get to that MPS, it had:
            a. 13 times the peak linear acceleration – 52.3g;
            b. 22.6 times the peak rotational acceleration—4538.0 rad/s^2;

2. a high velocity head to board impact (1995-1996 season), but to get to that MPS, it had:
    a. 15 times the peak linear acceleration – 60.7g;
    b. 24.6 times the peak rotational acceleration –4921.7 rad/s^2;

3. and a medium velocity head to ice impact (2013-2014 season), but to get to that MPS, it had:
    a. 21.7 times the peak linear acceleration – 86.9g; and
    b. 36.8 times the peak rotational acceleration –7353.5 rad/s^2.

The dynamic response values and MPS in my Initial Declaration reflect an impact consistent with 45% MPS and do not overstate the MPS values for the described impacts.

ii. Dr. Panzer reports that "plopping" into a chair creates 4g of peak linear acceleration and 150 rad/s^2 of peak rotational acceleration, which he claims results in 15.5% MPS when using my method of analysis. In my Initial Declaration, I noted 6 examples of 15%-16% MPS:

1. A medium velocity shoulder impact to the head (2003-04 season), but to get to that MPS, it had:
    a. 5 times the peak linear acceleration –19.9g;
    b. 8.1 times the rotational linear acceleration—1211 rad/s^2;

2. A medium velocity shoulder impact to the head (2013-14 season), but to get to that MPS, it had:
    a. 5.5 times the peak linear acceleration – 21.9g;
    b. 10.4 times the peak rotational acceleration – 1562 rad/s^2;
3. A high velocity puck impact to the head (2013-14 season), but to get to that MPS, it had:
    a. 35.5 times the peak linear acceleration – 142g;

b. 83.7 times the rotational acceleration –12561 rad/s^2;

4. A low velocity head impact to the glass (1995-96 season), but to get to that MPS, it had:
   a. 4.6 times the peak linear acceleration – 18.3g;
   b. 11.2 times the peak rotational acceleration – 1676 rad/s^2;

5. A medium velocity head impact to the glass (1986-87 season), but to get to that MPS, it had:
   a. 5.7 times the peak linear acceleration –22.9g;
   b. 16.9 times the rotational acceleration –2531 rad/s^2;

6. A medium velocity head impact to the glass (1995-96 season), but to get to that MPS, it had:
   a. 5.8 times the peak linear acceleration –23g;
   b. 16.8 times the peak rotational acceleration –2527 rad/s^2.

iii. Dr. Panzer reports that a "forehead slap" creates 12g of peak linear acceleration and 300 rad/s2 of peak rotational acceleration, which he claims results in 10% MPS when using my method of analysis. In my Initial Declaration, I noted 4 examples of 10% MPS:

1. A low elbow impact to the head (1986-87 season), but to get to that MPS, it had:
   a. .775 times the peak linear acceleration—9 g;
   b. 2 times the peak rotational acceleration—603 rad/s^2;

2. A low elbow impact to the head (2003-04 season), but to get to that MPS, it had:
   a. 1.13 times the peak linear acceleration—13.6g;
   b. 2.8 times the peak rotational acceleration—835 rad/s2;

3. A low elbow impact to the head (2013-14 season), but to get to that MPS, it had:
   a. 1.2 times the peak linear acceleration—14g;
   b. 3.6 times the peak rotational acceleration—1081 rad/s^2;

4. A punch to the head (1986-87 season), but to get to that MPS, it had:
    a. .63 times the peak linear acceleration—7.6g
    b. 2.1 times the peak rotational acceleration—617 rad/^{2};

It's also worth noting that a "forehead slap" with these inputs is not an every day activity and is closer to a low energy impact to the head and as such likely resulted in a number of the symptoms reported by participants.

iv. Dr. Panzer reports that jumping rope creates 4.5g of peak linear acceleration and 55 rad/s^2 of peak rotational acceleration, which he claims results in 25% MPS when using my method of analysis. In my Initial Declaration, I noted 3 examples of 25% MPS:

1. A high velocity impact to the glass (1986-87 season), but to get to that MPS, it had:
    a. 3.9 times the peak linear acceleration—17.4g;
    b. 30.7 times the peak rotational acceleration—1687 rad/s^2;

2. A low velocity head impact to the ice (2003-04 season), but to get to that MPS, it had:
    a. 7.2 times the peak linear acceleration—32.2g;
    b. 52.7 times the peak rotational acceleration—2900 rad/s^2;

3. A low velocity head impact to the ice (2013-14 season), but to get to that MPS, it had:
    a. 6.9 times the peak linear acceleration—31g;
    b. 78.5 times the peak rotational acceleration—4320 rad/s^2;

The dynamic response values and MPS in my Initial Declaration reflect the head impacts consistent with the reported MPS values. Dr. Panzer's methods of calculating MPS are unclear, but as one can see his claimed MPS values

for mild impact activity are grossly inflated when compared to the results of my study, and to those reported in scientific literature for similar events.[7]

c. Third, the vague description of how Dr. Panzer arrives at the MPS values makes it impossible to validate how he arrived at the extraordinarily, unexpectedly high MPS values set forth in his table. Further, it is unclear why the skull kinematics were constrained to the sagittal plane, particularly where the movement of the head is not restricted to the sagittal plane, and because such a restriction is uncommon in recording similar measurements.

d. Fourth, in generating the kinematic numbers from which the results were derived, Dr. Funk reported a variety of symptoms experienced by the subjects associated with these described low energy impact events to include jaw discomfort, minor head ache, ringing in the ears, minor stinging, momentarily dazed, head ache, forehead hurt, neck soreness, neck pain, stiffness, lower back soreness, sharp back pain, quadriceps soreness[8]; many of these are symptoms characteristic of a concussion diagnosis. This illustrates that while the events themselves were described as benign, they did result in symptoms associated with concussion.

e. Fifth, there is no reported MPS from scientific literature cited as to understood values for the four activities analyzed to confirm the MPS he found. If, in fact, the MPS values that were determined were consistent with MPS values found in the literature, then those values set forth in the literature for these every day activities should have been included for purposes of verification.

f. Sixth, a review of the acceleration values from my Initial Declaration demonstrates that I did not inflate the MPS values; but rather, Dr. Panzer's

[7] *See supra* n. 4.

[8] Funk JR, *et al.*, *Head and Neck Loading in Everyday and Vigorous Activities*, 39(2) ANNALS OF BIOMEDICAL ENGINEERING, 79-89 (2012).

methods for reconstructing Dr. Funk's data make the values artificially high and not consistent with reported MPS values for those activities set forth in literature. MPS values for activities noted in scientific literature[9] are far more closely aligned with my MPS results.

g. Seventh, the MPS values arrived at in my Initial Declaration are consistent with the peak acceleration values, which are consistent with the impact characteristics.

10. Those retained by the NHL do not offer any alternative factual findings as to what an analysis of the video produced show. If, as is claimed, my analysis was so flawed as to produce erroneous findings and conclusions, that would be shown by conducting an analysis in the manner advocated by those hired by the NHL. However, to my knowledge, none of the NHL's consultants conducted a single impact reconstruction using the measurements we took from the video using any impact reconstruction methodology that would purportedly be more accurate. Furthermore, none of the NHL's consultants conducted any finite element modeling using a finite element model that they claim would be more accurate. At the NHL's request, my lab spent more than four days processing and providing terrabytes of finite element modeling data so that their expert could feed the data into a model he deemed more appropriate. None of the NHL's declarants reported doing anything with the data, let alone demonstrating any significant difference when running the data through a different model.

Finite Element Modeling

11. In conducting my analysis of the frequency, speed, and strain of hits to NHL players' heads, I used a finite element model to determine the impact to the brain from impacts to the head. All finite element models are, as their name suggests, models and as such, have certain limitations. However, a finite element model such as the University of College Dublin finite element model (hereinafter the "UCD") that I used to come to my

[9] *See supra* n. 4.

conclusions, provides strain values that account for an array of impacts and assist my understanding of the effects of the observed impacts to the head.

12. One of the primary reasons that I used the UCD model, as opposed to other finite element models, is that I have a populated data set of more than 600 documented head impacts, with known outcomes, primarily involving head injuries in sport using the UCD finite element model, which provides an extensive data reference set. This data set includes approximately 120 ice hockey head impacts, including elite ice hockey levels, and provides an important reference against which to compare the MPS as determined in my Initial Declaration. This comparative data set was not only verified in using diagnosed concussions (*i.e.*, the "known outcomes"), but it was also verified by running that same data set through another finite element model—the Wayne State model. That data set of diagnosed concussions provides the information essential to verifying the accuracy and validity of the head impact measurements that my laboratory measured in relation to the NHL players. Without having a data set against which to compare results, a finite element model's accuracy is less able to be affirmed. None of the NHL's declarants discuss the vital importance of a verified data set, an omission that I deem unreasonable when considering the accuracy of an FE model.

13. The individuals retained by the NHL to critique my Initial Declaration also criticize my use of the GWV variant of the UCD finite element model. The reason that I use that variant is for consistency with the data set of over 600 impacts. In the event that I chose to use a different variant or no variant at all, the comparison to the referenced data set would not be feasible.

14. The finite element model that I employed in reaching my conclusions was a linear viscoelastic model. This type of model has the advantage of accounting for the full-time acceleration signal for the 3 linear and 3 rotational acceleration components resulting from head impacts. The linear viscoelastic UCD model also provides the effect of the magnitude and duration of the acceleration on the brain tissue, an important component to reconstructed head impacts in ice hockey. The magnitude and duration characteristics of the head impacts experienced by NHL hockey players are affected by the impact mass,

location, direction, compliance which vary a great deal when considering the events that represent head impacts in ice hockey, puck impacts, elbow impacts, shoulder impacts, punches, impacts to the ice, impact to the glass and impacts to the boards. These impacts represent a wide range of impact magnitudes and durations; therefore, using a model with viscoelastic properties like the UCD model provides strain values that account for the wide range of head impacts. Moreover, the linear viscoelastic model has the data set for comparison as previously described. Employing a different model, such as a hyperelastic model without appropriate or extensive reference data would be of little use in understanding the level of trauma represented by the head impacts in the Initial Declaration.

15. My Initial Declaration has been criticized based on the assertion that head trauma finite element models are only experimental research tools and should not be used for the purpose of "diagnosing" injury. I have not suggested that the finite element model diagnoses injury. Part of my assignment was to determine the levels of strain at which permanent cellular changes occur in the brain. This is not a "diagnosis" of an "injury." My understanding is that diagnosed brain injury requires a manifested impairment of cognitive or physical function. Finite element models are tools that can report the level of strain a certain impact produced. They are, as their name describes, models and research tools and as such have certain limitations. Finite element models are a means of treating data files to understand and interpret their characteristics in light of the question being posed. The models are representations of the brain and provide unique information, but must be compared to a data set for interpretation.

16. When, in my deposition, I indicated that the limitations that are inherent in finite element models were not included in my expert report, though they would be in a scientific article, I was speaking about what experts in the fields generally know the limitations to be, not about the accuracy of my Initial Declaration or the rigor with which I approached my analysis.

17. The suggestion by those retained by the NHL that the UCD finite element model is outdated appears to be meant to imply that the information reported in the Initial

Declaration is not true; this is not the case. While more recent models that purport to be more sophisticated in certain respects are attractive in that they intend to reflect more characteristics of the brain, they are limited by the displacement validation data to which all finite element models of the brain are subject, as Dr. Panzer appears to agree. The important advantage of the UCD finite element model used, which those retained by the NHL unreasonably ignore, is that it has the hundreds of concussive and non-concussive impacts in sports, including ice hockey, that have been analyzed using that model; this provides a very large and stable reference to support the opinions in my Initial Declaration and this Declaration. No finite element models, whether hyperelastic, anisotropic, or any of the other theoretically more "sophisticated" models have this reference data base size or ability. Their superiority is merely theoretical, and until they are verified by a robust data set such as the one supporting the UCD model, they remain in my view inferior for purposes of calculating strain values.

Sufficient Accuracy of Kinovea and Video Analysis

18. The individuals hired by the NHL to criticize my findings appear to have gone to all ends to find any possible or theoretical issue with my methodology and results, including suggesting that the Kinovea video software was insufficient and the video could have been broken down into further elements. The criticisms are not substantive and have no real effect on my ultimate findings and conclusions. We know that these items had no real effect on the results because the results are verified by being close to the results of other studies, as well as the robust, populated data set discussed earlier. In other words, we are confident in the methodology we employed with respect to our use of the Kinovea software because the results have been repeatedly validated—hundreds of times—through the known data set and the published literature. One manner of determining the validity of a data set is to frame it within the scientific literature on the topic to see if the data set is sound. The results, both of the data leading to the conclusions in the Initial Declaration, as well as other studies of head impacts, are proximate to the results set forth in scientific

literature and as a result, I am confident that the manner in which those results were derived, including the use of Kinovea, is scientifically sound.[10]

19. As to the specific issues cited by those hired by the NHL pertaining to Kinovea and video analysis:

a. The NHL playing surfaces are more than adequate to apply Kinovea grid. In the NHL, the playing surfaces are managed by a set of rules that define the dimensions of the rink and the markings on the rink so that they are uniform. Ice rink dimensions are published and were used to establish the dimensions for the ice surface markings; 2 goal lines, 2 defensive zone lines, center line, 5 face off circles, five face off dots, as well as the dimensions of the 2 goals, boards and glass. The ice is perfectly horizontal (frozen water) and provides an ideal horizontal plane of reference. These reference points work well for establishing known displacements to establish the grid. As to the concern that players' heads don't move along the gridded surface or are too distant to be accurate, we drew a line from the player's head perpendicular to the ice surface to accurately measure the horizontal displacement of the head in each frame. The game of hockey has players gliding along parallel with the ice allowing for accuracy in measures of displacement of the head.

b. The NHL's playing surfaces are not of such a character that scaling algorithms can't be accurately applied. As described in the preceding paragraph of this Declaration, the NHL ice hockey rink provides numerous references for establishing the grid used in the Kinovea program. Once the distances were established, they were confirmed using in-lab reconstructions. Each velocity was calculated twice with the average taken as reconstruction velocity; on those rare occasions when the velocities of the two measurements differed by more than 1 m/s, a third calculation was completed

[10] Post A, Koncan D, Kendall M, Cournoyer J, Clark JM, Kosziwka G, Chen W, deGrau S, Hoshizaki TB, *Analysis of speed accuracy using video analysis software*, SPORT ENGINEERING (2018), in press.

and the average of the two closest velocities was taken – this occurred in only four of all reconstructions.

c. Impacts in areas that were partially occluded were excluded whenever possible. However, where there were low numbers of videos for a particular category, we would use the method set forth in the preceding paragraph to make sure that the measurements obtained were valid.

d. In choosing video events for reconstruction, we used the following criteria in an effort to obtain the best and most valid events to obtain impact velocities using Kinovea:

   i. Clarity of the impact (non-obstructed view);
   ii. Proximity of the calibration grid to the impact;
   iii. Size of the calibration grid;
   iv. Orientation of the calibration grid (width vs length);
   v. Impact perpendicular to camera plane;
   vi. Quality of the reference dimensions; and
   vii. Event centered in the field of view.

e. The fact that the video of the impacts was down-sampled from 30 frames-per-second to 25 frames-per-second does not provide a measurable difference in accuracy. The reason is that the velocity of human movement is relatively slow and in this report ranged from between 1.53 m/s to 7.63 m/s. These velocities are easily captured using videos down-sampled to 25 frames per second.

f. Similarly, while de-interlacing the video footage does increase the number of frames available for analysis, the rate of human movement velocity does not warrant de-interlacing video footage. In this case, increasing the frame rate by de-interlacing video would not provide a meaningful increase in accuracy.

g. Lens distortion is a result of taking measurements at different locations on the lens with the greatest amount of distortion occurring at the edges of the

lens. As to the calculations in my Initial Declaration, the event primarily occurred in the center, or close to the center, of the lens. The minimal effect of lens distortion in the data collection did not warrant the use of a software to remove lens distortion.

h. As to the potential effects of panning and zooming, each frame was treated as an individual frame and analyzed with the appropriate reference and calibration procedure, which removed any such effect. Furthermore, the events for the analysis were chosen using the criteria set forth in paragraph d above, which criteria helped eliminate the occasions that were most susceptible to those effects.

i. The video analysis was conducted with care to ensure the velocity values used in the reconstructions were accurate.

20. During my laboratory's analysis of the video set produced by the NHL, any errors that arguably resulted from the Kinovea and video criticisms levied by the individuals hired by the NHL were minimized by internal procedures. Specifically, when a measurement appeared to be an anomaly, the measurement would be retaken; if the second measurement still appeared to be anomalous, a third, more senior, person would retake the measurement to ensure that it was correct.

21. The individuals hired by the NHL to criticize my findings and conclusions indicate concern about the ability of Kinovea to accurately account for measurements of velocity, suggesting that the movement of the head by a hockey player has too many different movements across different planes. Those concerns are exaggerated and do not result in my findings or conclusions being inaccurate. The measurements of velocity are not derived from measuring a hockey player as they skate down the entire length of the ice. Rather, velocity is measured in a very short time frame, specifically within .20 seconds prior to impact until .15 seconds after; the brief nature of the period of measurement eliminates any effect of "bobbing" or "weaving" of the head. Humans actually move, relatively speaking, very slowly. To change the direction of a 10 pound head in .2 seconds requires an impact. Head impacts in hockey occur over 15 milliseconds (.015 sec).

Therefore, the suggestion that a player skating at 5-13 meters/second impacting the glass with their head can decelerate their head during the last .08 seconds without an impact is not realistic.

22. The only two measurements that are taken in relation to use of the Kinovea software are location of the hit and velocity. The individuals hired by the NHL suggest that the velocity measurement is incorrect and is overstated. However, as previously mentioned, I have performed hundreds of reconstructions over the course of eleven years. While there will be errors, as is true in any study, I have done a sufficient number of reconstructions with internally consistent results. Combined with what is consistently reported in the related literature, this experience and historic results demonstrate that the methods, facts and conclusions set forth in my Initial Declaration are scientifically sound.[11]

23. The impact velocities used in the impact reconstructions that resulted in the conclusions of my Initial Declaration are well within the expected impact velocities reported in the scientific literature and if anything are conservative and not unrealistically high, considering the following:

a. An elite ice hockey player can skate up to 13 m/s[12] and plays the game at as high a skating velocity as possible in order to gain an advantage over their opponent. Skating velocity is an important and valuable asset in the game of ice hockey. NHL ice hockey players represent the very best hockey players in the world. It is widely accepted that skating is considered a foundation skill of the game and in order to succeed to the highest level all NHL players have to have the highest level of skating skill. While there are some limitations concerning the methods obtaining impact velocities from video it

[11] *Id.*

[12] Ken Campbell, *NHL Players Keep Getting Faster, Stronger, and More Skilled—How Far Can Hockey Evolution Go*, THE HOCKEY NEWS, Sept. 14, 2015, available at http://www.thehockeynews.com/news/article/nhl-players-keep-getting-faster-stronger-and-more-skilled-how-far-can-hockey-evolution-go (noting that Mike Gartner skated 570 feet in 13.386 seconds rendering a speed of 12.979 m/s).

is important to consider the impact values in light of reported velocities in the scientific literature. For elbow and shoulder impacts, the highest impact velocities used in reconstructions in this report was 7.0 m/s (54% of 13 m/s skating velocity) and 6.31 m/s (48% of 13 m/s skating velocity) respectively, well below the maximum skating velocity of 13 m/s.

b. Slap shots in hockey have reported puck velocities as high as 48.5 m/s[13], shooting the puck at high velocity is an important part of success and a common event in the highest level of hockey in the world (NHL). The puck impact velocity of 22.68 m/s (47% of 48.5 m/s) used in the reconstruction for the high velocity puck impacts in my Initial Declaration is very conservative and would be a common event in an NHL hockey game;

c. For a punch, the literature reports high velocity hand to head impacts to be as high as 11.9 m/s for boxers.[14] 7.16 m/s (60% of a boxer's punch of 11.9 m/s) was used for the reconstructions for the high velocity punch impact in my Initial Declaration. While fighting is penalized in the NHL, it is a common occurrence in the game and professional hockey players train to fight and know how to punch;

d. For head to head impacts, which includes two moving heads, I used a velocity of 5.35 m/s (41% of 13 m/s skating velocity) to reflect those high velocity impacts; again, this is well below high skating velocities of 13 m/s.

e. Head impacts to the ice can be as high as 6.15 m/s for a falling 6 foot 2-inch player[15]; however, the highest reconstruction for falls in the reconstructions

[13] *Id.* (noting Shea Weber won the hardest shot competition with a blast of 108.5 miles per hour).

[14] Walilko T., Viano D., Bir C., *Biomechanics of the head for Olympic boxer punches to the face,* 39(10) BRITISH J. SPORTS MEDICINE, 710-719 (Oct. 2005).

[15] Calculated for a 6' 2" player with 2" ice skate blades.

in my Initial Declaration was calculated as 5.43 m/s, (88% of maximal falling velocity) well below the possible high impact velocity for a fall.

f. The highest impact velocity for impacts to the glass was 5.47 m/s (42% of 13 m/s skating velocity) and for impacts to the boards 7.60 m/s (58% of 13 m/s skating velocity); again, this is well below the skating velocity of an ice hockey player 13 m/s.

g. The game of ice hockey is played as a contact sport at very high velocities (up to 13 m/s skating velocity) on a very slippery surface (ice) encompassed by hard ice, boards and glass. The game includes high puck velocities (48.5 m/s), aggressive body contact (13 m/s skating velocity), falls (6.15 m/s) and fighting (punches of 11.9 m/s) all contributing to head impacts at all levels of energy. The average head impact velocities used in the impact reconstructions in this report include:

   i. elbow impacts, 3.13 m/s for low, 5.49 m/s for med and 7.0 m/s for high;

   ii. shoulder impacts: 2.53 m/s for low, 4.42 m/s for med and 6.31 m/s for high;

   iii. head to head impacts: 2.11 m/s for low, 3.54 m/s for med and 5.35 m/s for high;

   iv. punch to head impacts: 2.42 m/s for low, 4.44 m/s for med and 7.16 m/s for high;

   v. puck to head impacts: high 22.68 m/s;

   vi. glass impacts: 1.90 m/s for low, 3.1 m/s for med and 5.47 m/s for high;

   vii. head impacts to board: 2.16 m/s for low, 4.30 m/s for med and 7.60 m/s for high;

viii. head impacts to the ice 1.86 m/s for low, 3.71 m/s for med and 5.43 m/s for high velocity impacts.

The impact velocities used in the impact reconstructions resulting in my Initial Declaration are well within the expected impact velocities reported in the scientific literature and are, if anything, conservative.

24. Dr. Phil Rousseau completed his Doctorate and research under my supervision in our lab; we followed the methods described in his research with minor changes that allowed us to include the velocities experienced by the NHL players.

Scope of Conclusions

25. In order to come to a conclusion concerning the frequency of head impacts to an average NHL player in a game that caused injury to their brain tissue, it was necessary to have a strain level criteria at which that risk occurred. To determine the point at which the risk for damage to brain cells begins, I used the impact level measured in MPS at which strain begins to effect brain cells and their function. I relied on publications in scientific literature for those criteria as noted throughout my Initial Declaration.

26. Neither in my Initial Declaration, nor in this declaration, am I contending that at 5% MPS, there will be permanent "brain injury" to an individual. Rather, I am using the strain measurements from the impact reconstructions and then applying the information set forth in scientific literature and by other experts in this litigation regarding the point that morphological changes begin to take place on a continuum of injury that begins with functional impairment and morphological changes and continues into morphological cell damage; that continuum begins at strain levels as low as 5%.[16] I acknowledge that not

[16] T. Yuen, et al., *Sodium channelopathy induced by mild axonal trauma worsens outcome after a repeat injury*, 87 J. NUEROSCI. RES. 16, 3620-25 (2009) (providing that at strains of 5%, "undulations typical of axon injury were … present in all suprathreshold populations"; this article was cited in my initial Declaration at p. 11 footnote 13, and p. 19, footnote 46. *Compare* Expert Report of Robert C. Cantu ¶ 42 (describing the effects of internal chemical reactions in the cell, including the allowance of calcium in which leads to dysfunction and can activate enzymes that eventually lead to cell damage or death) *with* T. Yuen, *et. al*,

every change at the 5% level is permanent. However, the literature indicates that there is a strong connection between strain and damage to white matter on a continuum of damage to brain tissue, which includes permanent injury to that tissue. That is the literature that created the baseline against which I applied the measurements from my impact reconstructions and created the data set in my initial declaration.

27. The NHL and the experts seeking to discredit my testimony appear to have significant concerns regarding the 5% MPS as the threshold, despite the scientific literature demonstrating functional impairment of the cell at that level, as well as undulations typical of axonal injury. However, in many ways the 5% threshold is irrelevant because the lowest MPS from the reconstructed impacts was 8% MPS (and this was a single impact). All the rest of the impacts were 10% MPS or greater. The MPS results from the reconstructions are sound and to the extent there were different thresholds applied to determine damage to the brain tissue, it would simply change the number of games an average NHL player would play before receiving an impact sufficient to create a risk to likely have permanent brain tissue damage. As examples:

a. The 8% strain threshold, reported in the research literature as the beginning of conduction block in a study of spinal nerve roots,[17] could be used. The average number of games an NHL player would have to play before receiving an impact sufficient to reach the 8% MPS threshold would be .99 games[18]—

supra (describing that even at mild strain rates, increases in calcium induce axon degeneration).

[17] A. Singh et al., *A new model of traumatic axonal injury to determine the effects of strain and displacement rates*, 50 STAPP CAR CRASH JOURNAL, 601, 609 (2006), cited in my Initial Declaration on page 19, footnote 46.

[18] The number of games an NHL player would play in order to be subject to an impact at the given threshold (*e.g.* 8%, 10%, 14%, 21%) was calculated by: reviewing Table 5 of my Initial Declaration to determine which types of events were below the given threshold; then removing those sub-threshold events from Tables 2-4; then taking the total number of remaining events (supra-threshold) and dividing it by the total number of games (120) by the total number of players (38) and taking the reciprocal.

the same number of games for the 5% threshold because all impacts measured at 8% or more MPS.

b. The 10% strain threshold, reported in the research literature to be the level at which there was evidence of torn axonal fibers concomitant with observed functional injury and at which macroscopic hemorrhaging often resulting in cell death[19], could be used. The average number of games an NHL player would have to play before receiving an impact sufficient to reach the 10% MPS threshold would be 1.11 games[20].

c. The 14% strain threshold, reported in the research literature as a conservative threshold for morphological damage to white matter, [21] could be used. The average number of games an NHL player would have to play before receiving an impact sufficient reach the 14% MPS threshold would be 1.55[22] games.

d. The 21% strain threshold, reported in the research literature to be the optimal threshold for morphological damage to white matter,[23] could be used. The average number of games an NHL player would have to play before receiving an impact sufficient to reach the 21% MPS threshold would be 4.35 games[24].

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[19] A. Singh *et al.*, (2006) *supra* at 619.

[20] *See supra*, n. 18.

[21] A.C. Bain & D.F. Meaney, *Tissue-level thresholds for axonal damage in an experimental model of central nervous system white matter injury*, 122 J. BIOMECH. ENG. 6, 615, 620 (2000), cited in my initial declaration at p. 7 n 1, and p. 10 n 11.

[22] *See supra*, n. 18.

[23] Bain & Meaney, *supra* at 620.

[24] *See supra*, n. 18.

The point is that the outcome in the overall numbers and conclusions in my report do not significantly change as the result of using alternative strain thresholds. Given the conservative nature of my approach in collecting data, in that not all hits in NHL games were observed by the camera[25], it is my opinion that permanent cell changes would have occurred as a result of a player participating in a single NHL game.[26] However, even if one were to apply 21% MPS as the threshold, anyone who played an average of 4.26 or more NHL games would be expected to have experienced permanent cell damage.

28. My work as a consultant in the sports equipment industry has in part focused on the design of headwear to reduce, and where possible eliminate, the brain tissue damage to an athlete as the result of impacts.

29. During the course of the aforementioned work, as well as my academic work, the thresholds for trauma to brain tissue has been a necessary and important point of data. Understanding the thresholds has been an integral part of that work.

30. The information concerning the level of MPS at which there is damage to white matter has come from studies that admittedly use the anatomy of animals. As is the

[25] Wilcox, BJ, *et al*, *supra* n. 2.

[26] In my Initial Declaration, I concluded that an average NHL player likely received a head impact in each game sufficient to cause permanent injury to brain tissue. (Initial Decl. ¶ 65.) In preparing for my deposition, I discovered a miscalculation of the tables in my Initial Declaration, which I disclosed at the start of my deposition—I had divided certain figures by the number of players on one team in a game (19) versus both teams (38). (Hoshizaki Dep. T. 11:4-22 – 12:1-9.) I clarified that the effect of the change was that the lowest number of IPG (the 1986-87 season in Table 8 of my Initial Declaration) was not 1.19, but rather .6. (*Id.* 12:21-22 – 13:1-6; *see also* Hoshizaki Decl. at 33, Table 8; Hoshizaki Rebuttal Decl. ¶ 31.) I have now had the opportunity to properly calculate the data and put figures into Table 8 using 38 players and determined the impacts per game accordingly. (Hoshizaki Rebuttal Decl. ¶¶ 27, 31, Ex. A.) In responding to opposing counsel's questions during the deposition, I explained that the consequence of the miscalculation in relation to the lowest IPG in Table 8 of my Initial Declaration (the 1986-87 season) was that if using that figure, a player would be expected to experience an impact at the 5-8% MPS level more than once every two games. (Hoshizaki Dep. T. 12:21-22 –13:1-6.) However, when the number of impacts per player per game are averaged across all seasons, the average player experienced an impact of 5-8% MPS on average in a single game. (*See* Ex. A.)

case with countless scientific studies, the information in the literature concerning prospective head trauma research cannot ethically be performed on humans. That said, case studies, coupled with animal studies on the effect of white matter, show associations for humans reinforced by causation among animals; the difference at the neuron and cellular level between animals and humans is considered in this Declaration.[27]

31. In reviewing my Initial Declaration that I signed on November 27, 2016, there are certain corrections that I need to make, specifically:

a. In Tables 8 and 9 on pages 33-34 of my Initial Declaration, the estimated impact count per player should be for 38 players (2 teams of 19) and therefore:

i. On Table 8, the figures in the columns of Impacts/player for each season should be divided by 2 in order to get an accurate count. For example, for the 1986/87 season in Table 8, the impacts/per player for each level of impact velocity should be: .36 for Low, .22 for Medium, .02 for High, and .6 for Total. *See* Exhibit A for the table as it should have been set out.

ii. On Table 9, the figures in the columns of Hits/player should be divided by 2 in order to get an accurate count.

iii. The statements in Paragraphs 61 and 62 of my Initial Declaration should likewise be corrected with updated figures.

b. In Paragraph 59 on pages 26-27 of my Initial Declaration, the fourth and fifth sentences should instead read: "The reconstructed head impacts for the low velocity impacts primarily resulted in MPS between 10 and 13% with the

[27] *See* A. Singh, *et al.*, *supra* at 617 ("Spinal nerve roots are well suited for this study, because the structural environment of their axons is comparable to axons in the brain white matter and the size of the nerve root allows dissection and histological studies to be undertaken with relative ease"); Bain & Meaney, *et al.*, *supra* at 621 ("[e]vidence suggests that the mechanical behavior of central nervous system tissue does not vary significantly from species to species, so that strain-based injury criteria may be very similar between species").

exception of head impacts to the boards (18% MPS) and head impacts to the ice (24% MPS). Most notably, nearly all of the reconstructed head impacts significantly exceeded the reported minimum threshold (between 5% and 8%) associated with white matter damage and increases to tau and NFLP in CSF, with a vast majority of impacts greater than 10% MPS."

I declare under penalty of perjury that the foregoing is true and correct.

Dated: Feb 9, 2018

Thomas Blaine Hoshizaki, Ph.D

EXHIBIT A

TO REBUTTAL DECLARATION

OF THOMAS BLAINE HOSHIZAKI, PH.D

TABLE 8

5-8% Strain

5-8% THRESHOLD DAMAGING IMPACTS/GAME												
	1986/87			1995/96			2003/04			2013/14		
Impact Velocity	30 games	Per game	Impacts/ player	30 games	Per game	Impacts/ player	30 games	Per game	Impacts/ player	30 games	Per game	Impacts/ player
Low	406	13.53	0.36	659	21.97	0.58	785	26.17	0.69	963	32.1	0.84
Medium	252	8.4	0.22	165	5.5	0.14	531	17.7	0.47	636	21.2	0.56
High	22	0.73	0.02	20	0.67	0.02	104	3.47	0.09	82	2.73	0.07
Total	680	22.66	**0.6**	844	28.14	**0.74**	1420	47.34	**1.25**	1681	56.03	**1.47**